AN 11-MONTH-OLD, previously healthy boy, was admitted after a 3-day history of daily fevers up to 38.8°C. The patient was otherwise active and playful and had no history of rhinorrhea, cough, vomiting, or diarrhea. On admission, his temperature was 38.8°C, but he was alert, well nourished, and in no respiratory distress. His chest was clear on auscultation, and the findings from the remainder of his examination showed no abnormalities. The white blood cell count was 20.7×10^9/L, with 0.61 segments, 0.06 band forms, and 0.29 lymphocytes, and 0.04 basophils. The hemoglobin level was 98 g/L (reference range, 140-175 g/L); hematocrit 0.28 (reference range, 0.41-0.50); and a platelet count of 0.001×10^9/L. A chest radiograph done on admission, with the patient in the right lateral decubitus position, is shown (Figure 1). Blood culture showed no growth of organisms after 72 hours, and purified protein derivative for tuberculin was non-reactive with a positive control.

The patient was treated with intravenous cefuroxime, but continued to have fevers, even though the antibiotics were changed to a combination of nafcillin sodium and cefotaxime sodium. On day 6, he developed tachypnea and a fever of 40°C. Another chest radiographic scan (Figure 2) and a thoracentesis was performed, which removed 100 mL of cloudy, bloody fluid. Fluid analysis showed protein at 43 g/L (reference range, 60-80 g/L); lactate dehydrogenase, 3133 U/L; and a pH of 7.1. Gram stain was negative, and culture showed no growth of organisms after 72 hours. Acid-fast bacilli stain and culture were also negative for organisms.

The patient underwent a video-assisted minithoracotomy, lung decortication, and pleural drainage. Findings are shown in Figure 3. After surgery, he was afebrile while he was being treated with intravenous antibiotics and was given oral antibiotics when he was discharged from the hospital 10 days later. Evaluation results for immunodeficiency status was negative, but a swallowing study showed significant aspiration of formula when mixed with contrast material given by bottle.
Lung Abscess With Rupture Into the Pleural Space

Figure 1. Chest radiograph of the patient in the right lateral decubitus position shows a left lower lobe pneumatocele, 4-cm in diameter, with an air-fluid level.

Figure 2. Frontal chest radiograph shows a left pleural effusion containing multiple air bubbles and marked diminution in size of the left lower lobe pneumatocele.

Figure 3. View of the left lung taken at the time of the surgery demonstrates the cyst (arrow) that had ruptured into the pleural space, fibrinous adhesions, and hyperemia of surrounding tissue.

A lung abscess is a localized infection with central necrosis and suppuration of the lung parenchyma, surrounded by a thick wall of infected and inflammatory tissue. This process may establish communication with an airway and cause partial expectation of the purulent content and a resultant air-fluid level. Lung abscesses are categorized as primary (those occurring in otherwise healthy children) and secondary (those which occurring in patients with predisposing factors such as cardiopulmonary diseases, immunodeficiency or immunosuppression states, prematurity, recurrent aspiration, or cystic fibrosis).1

The overall incidence of lung abscess in children is low compared with the preantibiotic era and estimated to be 0.7 per 100,000 admissions per year.2 A wide array of microorganisms, including bacteria, fungi, and parasites are responsible. Staphylococcus aureus is the organism most frequently isolated, followed by Streptococcus pneumoniae, Haemophilus influenzae, Mycoplasma pneumoniae, Pseudomonas aeruginosa, and Klebsiella pneumoniae. Thirty percent of lung abscesses are due to mixed organisms,2 and anaerobes are commonly found, especially in abscesses resulting from aspiration1; however, determination of the origin also depends on the method by which the microorganism is obtained.4

Children with lung abscess present with a wide variety of symptoms including fever, malaise, weight loss, cough, vomiting, and tachypnea. Tachypnea, sternal retractions, diminished breath sounds on the affected side, and respiratory crackles are common signs, although the physical examination may show no abnormalities.

Lung abscess can be diagnosed by a chest radiographic scan showing a thick-walled cavity containing an air-fluid level. When caused by aspiration, the abscess can occur in any part of the lung,3 with a predilection for the dependent regions. A primary lung abscess is almost always solitary, whereas secondary abscess can be solitary or multiple. An abscess located in the peripheral region of the lung often makes distinguishing between lung abscess and loculated empyema difficult, and may require ultrasonography6 or a computed tomographic scan of the chest.7 In addition, mediastinal and hilar lymphadenopathy may be observed.8 Percutaneous transthoracic needle aspiration is the most accurate and safe procedure to determine the infectious origin.5 Alternative methods include bronchoalveolar lavage, transtracheal aspiration, airway brushing, and sputum cultures.

Conservative treatment of primary lung abscess due to S aureus with intravenous antibiotics has resulted in complete clinical and radiologic recovery, and normal, long-term pulmonary function.7 Duration of treatment varies from 4 to 6 weeks total, with an initial treatment period of 2 to 3 weeks with intravenous antibiotics.2 Empirical antibiotic coverage for S aureus is recommended in all primary lung abscesses, and antibiotic coverage for anaerobes (penicillin, ticarcillin/clavulanic acid) should be considered for abscesses suspected to be secondary to aspiration pneumonia. Gram-negative coverage (ticarcillin/clavulanic acid, gentamicin sulfate, tobramycin) is considered in hospitalized or debilitated patients.10

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